PFT RESULTS:

Chart #1 - Pulmonary Function Results (PFT)

Category	No.	% of Total	Comments
Total cases	1691	100%	All claims derived from random selection of 22,578 claims submitted under agreements compensating for impairment
impaired	225	13.3%	Fulfill ATS requirements and contractual criteria
impaired deficient	106	6.26%	Additional cases which failed to meet ATS criteria and potentially could qualify if retested and ATS criteria was met*
unimpaired	886	52.33%	Unimpaired according to NSP criteria. 118 of those had no tracings
	282	16.67%	No PFT performed*
	98	5.8%	Flawed TLC
	94	5.55%	Misc. 72 due to PFT performance, 21 due to failure to provide ILO or missing data, illegible, etc.
Totals	1360	80.36%	Total unimpaired

* likely to provide additional impaired cases if retested. See Methodology re: deficiencies.

Chest x-ray:

In my opinion the underlying x-ray reports were problematic.

1) Only five B readers accounted for over 80% of the claims filed (Chart 2A)

2) The spectrum of asbestos related nonmalignant disease reported by these five "B" readers was inconsistent with that anticipated from the peer review literature authored by other plaintiff experts. The literature documents pleural findings to be far more prevalent than parenchymal. (Chart 2B)

3) There was a high degree of inter-reader variability among these five "B" readers (Chart 3)

4) There was a high degree of inter-reader variability when these five "B" readers findings were compared to over 40 other "B" readers or other physicians submitting plaintiff reports in this study. (Chart 2B)

5) Certain idiosyncracies were observed among these "B" readers which further raised substantial questions. Examples including a high prevalence of all six lung zones involved with low profusion by some readers and a high prevalence of en face plaques by another, etc.

6) The overwhelming majority of ILO interpretations were of low profusions as noted in the "ILO Profusion Chart". (Chart 2)

Additional information can be found in the Section on X-ray Review, Pages 15-23 and in the conclusion.

CHART #2

ILO Profusion Chart

A)	158	no profusion
B)	5	0/1
C)	639	1/0
D)	641	1/1
E)	155	1 / 2
F)	30	2/1
G)	42	?
H)	12	2/2

Chart 2A - "B" readers

"B" readers in USA (1994-1999)	-	600+
"B" readers accounting for > 80% of claims	-	5

Chart 2B Summary of X-ray Findings - Pleural/Pulmonary Ratios

Ratio Pl:Pulm*	Ratio Pl:Pl/Pulm	Ratio Pl only/Pulm/ Pl Pulm	Source
≥2:1	≥2:1	≥ 1:1	Peer review lit. (See Appendix 3)
1:35.3 (28/988)	1:8.7 (28/244)	1:47.6 (28/1332)	"B" readers (See Chart 3)
		1:2.46 (96/236)	43 other "B" readers or physicians retained as experts by plaintiff for rest of cohort (332 cases) (19.6% of cases)

* PL = Pleural
Pulm - Pulmonary
Pulmonary is used as parenchymal in this chart

Thus, over 97.5% either had no profusion reported or were 1/ 2 or below. Only 42 cases were reported 2/1 or 2/2 (2.5%). No higher profusion were reported. Lower profusion levels may be more problematic concerning their interpretation.

As the x-rays form the foundation for the diagnosis of nonmalignant impaired, unreliability in a substantial number of cases would put significant downward pressure on the 13.3% of the cases which fulfilled NSP criteria for impairment as shown by PFTs.

Estimate of Effect of X-ray Findings on Rate of Impairment:

In order to assess the impact the x-ray findings might have on the PFT results, I matched impaired PFTs with x-rays showing evidence of pleural disease. An estimate of the incidence of impaired claims which might be attributable to asbestos in this cohort is between **8.16%** (claims which are impaired without deficiencies on the PFTs) and **10.46%** (claims which are impaired including deficiencies) of claims submitted. If all impaired cases with deficiency are allowed and 7% of those without PFTs are presumed impaired, an estimate of **11.65%** is made. Further investigation of the cases through audit of the x-rays, review of occupational history and exclusion of other more probable cause may further reduce this number.

To arrive at these numbers, the following assumptions were used:

1) Any impaired case with pleural changes - regardless of magnitude including unilateral, A1, en face or unilateral diaphragmatic was accepted for this portion of the study. (Despite the fact the overwhelming majority did not meet NSP criteria for pleural disease).

2) This number was increased by 50%. This would then result in 1/3 of cases of asbestosis being "parenchymal only". This is the number sometimes cited as the frequency with which asbestosis occurs in the absence of pleural disease. It is acknowledged that this estimate may be slightly high as some "pleural only claims" are present in #1.

3) Any case meeting NSP criteria for impairment was considered asbestos related, when pleural disease of any type was present.

   A) impaired
   B) impaired with deficiency

4) Whenever there were discrepancies between ATS and NSP criteria for assessing pulmonary function studies, the method favoring the claimant (plaintiff) was chosen.

The following reflect impairment attributed to asbestos (nonmalignant):

   A) Impaired cases without any deficiencies with pleural disease -

(86 without calcification and 6 with calcification)	-	92
Add 50% to account for asbestotics without pleural disease	-	46
Total		138

$$\frac{138}{1691} = \mathbf{8.16\%}$$

B) Impaired cases (**including deficiencies**) with pleural disease

(112 without calcification, 6 with calcification)	-	118
Add 50% to account for asbestotics without pleural disease	-	59
Total		177

$$\frac{177}{1691} = \mathbf{10.46\%}$$

C) 282 cases without PFT - assume 7% impairment rate = 20 cases

D) Impaired cases computed by accepting all deficient and non-deficient impaireds with pleural change and adding 20 cases (7% of those having no PFTs)

177 (all impaired) + 20 = **197 (11.65%)**

Specific issues concerning the ILO and x-rays interpretations and their impact on this study are addressed in detail in the section x-ray review (pages 15-23). The x-ray peer review literature can be found in Appendix 3.

Specific issues concerning performance of pulmonary function studies (PFTs) and systemic or recurrent problems which materially affected determination of impairment are identified and addressed in Appendix 2 and the section on

Pulmonary Function Testing.

Future impaired claims (2000-2049) should further decline if proper medical guidelines are enforced.

X-RAY REVIEW

Purpose X-ray Review:

Under the terms of the settlement Agreements, future claims for impairment depend on the ILO scale for grading x-rays and utilize a **"sliding scale"** with **criteria for impairment being defined differently for pleural disease, 1/0 or 1/1 and greater.** Thus the **first step** in determining impairment is predicated upon a reliable radiographic interpretation. This issue is complicated by the fact that at low profusions (which represent the majority of cases submitted), there is some degree of subjectivity in the interpretation of the x-ray. In the evaluation of a single x-ray, there may be intra and inter reader variability. However, when large numbers of radiographic interpretations are submitted (such as in this cohort), it is possible to objectively compare the B reader's interpretations with findings of other large groups of asbestos exposed individuals reported in the peer review literature. The following represents a method for objectively assessing the ILO interpretations accompanying the 1,691 claims which were analyzed for impairment under Part I.

Methodology:

No x-rays have been submitted either for my personal review or to any other independent reviewer or panel of reviewers to the best of my knowledge. All x-ray reports were recorded as reported by plaintiffs expert . without challenge. I have performed a review of the asbestos literature emphasizing studies performed by authors who have either been retained as experts or are commonly recognized as authoritative by plaintiff counsel in asbestos litigation. Studies requested or commissioned by unions are also included. I have attempted to identify studies which are performed on individuals with high levels of asbestos exposure, low levels of asbestos exposure and various durations of exposure. Studies are chosen to represent various settings in which asbestos is present including shipyards, railroads, construction trades, insulators, petrochemical industry, and others. Because asbestos causes a spectrum of disease effecting both the pleura and the pulmonary parenchyma, it is possible to define the distribution of nonmalignant disease among the various cohorts which have been reported. The interpretation submitted by the plaintiffs "B readers" are then compared to the findings of the peer review literature. In addition, this methodology allows comparison of "B" readers with each other.

Summary of Asbestos Literature: There is no dispute that asbestos exposure causes a spectrum of benign and malignant disease affecting both the pleura and pulmonary parenchyma (either separately or with both present). This review has focused on the prevalence and distribution of nonmalignant disease detected on

chest x-ray in various populations. Regardless of degree of exposure, duration of exposure, occupation, industry (shipyard, railroad, construction, petrochemical), bystander or commercial; whether by x-ray review or pathologic assessment of asbestos fiber count, a distribution of disease along a spectrum is found. Pleural plaques (or fibrosis) are the most common finding and in most series are observed at least twice as often as parenchymal disease alone or combined pleural and parenchymal disease. Where there is lower dose exposure or exposure of shorter duration, the ratio of pleural to parenchymal cases is higher. Such exposure may be anticipated in the future.

These findings from the literature are compared to an analysis of the distribution or spectrum of radiographic findings of nonmalignant disease observed by the "B readers" in the 1,691 cases submitted. This analysis allows determination whether x-ray interpretations submitted for nonmalignant impaired claims are consistent with the findings of peer review literature whose authors are deemed to be experts or trustworthy by plaintiff counsel. Further, it allows identification of reading patterns of "B" readers and comparison between plaintiffs "B" readers. Examples of many of the articles from the literature are found in Appendix (3).

Results of B reader ILO report review:

1) **Dr. H** - of the 1,691 cases submitted, 772(46%) were reviewed by Dr. H (Exhibit X-l). Of these 772 claims only one patient (Mr. Elijah Doughty 490583) was described as having bilateral pleural disease in the absence of interstitial disease. 67 other patients had pleural disease accompanied by parenchymal disease. Of these at least 31 were unilateral with many being only unilateral diaphragmatic plaque or unilateral en face plaque. Another 16 were either category A-1 or otherwise minimal. Few met OC criteria for pleural disease. 1 patient, Rose Delmon, had unilateral pleural disease without parenchymal disease.

Dr. H had a propensity for diagnosing 1/0 or 1/1 interstitial fibrosis in all six lung zones, a pattern which is atypical for asbestosis with low profusion. (Exhibit X-4) is a small sampling to illustrate this phenomena. Dr. H had relatively few parenchymal cases with associated pleural findings and the virtual absence of "pleural only" cases is troubling. The latter serve as the benchmark by which other nonmalignant x-ray findings are gauged in this study.

2) **Dr. S** - total x-rays read 229. Total with pleural disease - 89, (9 of which were unilateral). Pleural disease with no interstitial disease - 19 . Parenchymal disease, no pleural 140. Both parenchymal and pleural - 70.

3) **Dr. K** - read 212 radiographs. He found pleural disease accompanying parenchymal disease in 138 (at least 18 cases were unilateral). There were **no cases** in which there was pleural disease without pulmonary disease present. There were 74 cases in which there was pulmonary disease but no pleural disease.

Dr. K also had a propensity for diagnosing 1/1 fibrosis in **all six lung zones**, a pattern which is not usually seen in the early stages of asbestosis.

Dr. K also had a propensity for finding obscuration of the right cardiac border as well as the left cardiac border. Such findings would usually occur with higher profusion of irregular opacities (2/2). However, Dr. K noted this abnormality in numerous cases where he reported 1/0 or 1/1. Examples include those of James Crenshaw (419-30-1151), William E. Clark (423-40-3590), Willie J. Crum (423-52-1022), Charles G. Jones (421-36-3731), James Eason (420-40-3954), and Roscoe P. Green (424-44-4227). Interestingly, I did not frequently find similar descriptions from the other plaintiff B readers. Examples are provided in (Exhibit X-5).

4) **Dr. L** - total number of cases 87. The number of pleural without pulmonary (1) (Lonnie Corbin -397475). The number of pulmonary without pleural 63, number with combined pleural and pulmonary 23 (at least 5 are unilateral pleural).

5) **Dr. JB** - total cases reviewed 60. Pleural disease was found in 53. Pleural disease without parenchymal disease was found in 6. Pleural disease and parenchymal disease combined in 47. Parenchymal with no pleural disease was found in 7.

> **Comment:** There is an unusual similarity between a significant number of Dr. JB's reports. Among the B readers he is unique in the propensity for finding en face plaque. This is worrisome since overlying pectoral muscle shadows, fat and other soft tissue may mimic en face plaque. As he described pleural findings in 53 out of 60 cases, with many of these pleural findings being "en face" and given the absence of similar observation by any other plaintiff B reader (and an uncommon finding in my experience), I view these reports with some caution. Enclosed is a copy of the computer print out of all 60 of Dr. JB's (Exhibit X-6) reports which were submitted. The abbreviation EF PLQ or PLQ EF indicates en face plaques in those instances. (See pages 3 and 6 of Exhibit X-6).
>
> Assuming that the cases did represent en face plaque there is still significant deficiency of pleural only cases vs pleural plus parenchymal cases.

General observation concerning B readers:

During the years that these radiographs were interpreted, there were over 600 readers in the United States. Assuming that the 1,691 cases submitted represented a true "national epidemic" of asbestos disease as suggested by the recent onslaught of claim submissions, it would be reasonable to assume that there would be a broad representation of physicians detecting such disease. Of the 1,691 claims submitted, 772 were submitted by Dr. H, 229 by Dr. S, 212 by Dr. K, 87 by Dr. L accounting for 1300 cases or **76.8%** of all claims. If Dr. JB is added, these five "B" readers account for 1360 **(80.4%)** of claims. Dr. H alone accounted for approximately 45.6% of all claims submitted in this study. Key to identity of "B" readers is present in (Exhibit X-8)

Because such a small number of physicians account for such a large percentage of the claims previously submitted, and because of the prominent role which ILO interpretation plays in the existing agreements, the need to objectively evaluate the reliability of their reports cannot be underestimated. A noteworthy example is that Dr. H (772) and Dr. K (212) had only 1 bilateral pleural case without parenchymal disease out of 984 claims.

Chart 3

Summary of five B readers:

Drs.	#X-ray	Pleural only	Parenchymal only	Pleural & parench	Total Pleural
H	772	1 bilateral 1unilateral	704	66	68
S	229	19	140	70	89
K	212	0	74	138	138
L	87	1	63	23	24
SB	60	6	7	47	53
TOTAL	1360	28	988	244	372

Based upon the "pleural only" cases reported by these five "B" readers, the number of claims reporting asbestosis (parenchymal only) or combined (pleural and parenchymal) far exceed that which would be expected based on the peer review literature (Appendix 3).

Summary of X-ray results (B reading and x-ray reports from 1,691 cases randomly selected from 22,578 claims alleging nonmalignant asbestos disease):

1) During 1994 to 1999 there were over 600 "B" readers in the United States

2) **76.8%** of the claims submitted resulted from "B" readings were generated by **4** "B" readers. When a 5th "B" reader was considered, **80.4%** of all claims filed were accounted for. One "B" reader Dr. "H" accounted for 46% of the claims.

3) The ILO and x-ray interpretations differ from what would be anticipated from a randomly selected panel of reliable "B" readers nationwide

4) Peer review studies consistently show an excess of pleural disease over pulmonary asbestosis in a variety of exposure settings.

5) **None** of the five "B" readers accounting for over 80% of the claim submissions found more "pleural only" cases than pulmonary asbestosis.

6) The readings generated by these "B" readers yielded results which were **not consistent with the spectrum of nonmalignant asbestos related disease identified within the peer review literature** authored by plaintiffs experts or authors deemed authoritative by plaintiff experts.

7) The reading pattern of certain "B" readers showed certain unique profiles such as finding all 6 lung zones involved in a low profusion x-ray and no pleural change. One reader was uniquely proficient at identifying en face plaques. Few calcified plaques were ever noted.

8) The reading patterns were not internally consistent among the 5 "B" readers accounting for 80.4% of the claims.

9) Of the 1691 cases only **124 "pleural only"** cases could be identified.

10) Dr. H (772), Dr. K (212) and Dr. L (87) reviewed a total of **1071** cases. **9** "Pleural only" cases (at least 1 being unilat.) were reported. The incidence of "pleural only" disease reported by these 3 doctors is only **(0.8%)**.

11)5 of the "B" reader accounted for **1360** cases (80.4% of cases). Of these only **28** were "pleural only" claims in the absence of pulmonary disease **(1.84%)**. The **ratio** of pulmonary asbestosis and pleural parenchymal to "pleural only" was **47.6:1.**

12) In the "remaining" 331 claims (19.6% of all x-rays) submitted by over 40 other "B" readers and physicians (Exhibit #8), 96 cases were "pleural only" claims (29%). The **ratio** of pulmonary asbestosis to "pleural only" claims was **2.46:1.**

An estimate of impaired cases attributable to asbestos:

The above raised questions concerning the reliability of a significant number of the underlying x-ray interpretations. This leads me to believe that the actual percentage of impaired nonmalignants was no more than and in fact, was less than the 13.3% rate computed for the cohort. To address this issue, I matched the x-ray findings with the PFT findings. Pages 12-14 reflect this analysis.

X-ray Discussion:

Explanations for variance noted in x-ray:

Possibilities include:

1) Reader error
2) Reader bias
3) Other more probable nonoccupational cause .heart failure, cardiac surgery, kidney failure, pneumonia, TB, autoimmune disease and others were noted on death certificates and IMEs
4) Other more probable occupational cause (coal mining, steel mills, foundry work, etc.)

It was noted that a large number of these claimants had previously worked in mines, sandblasting, foundries, steel mills or other trades where exposure to silica and fibrogenic dusts (other than asbestos) may result in abnormal radiographic findings. These diseases may have x-rays characterized by parenchymal disease without pleural disease and also have upper lobe involvement.

In reviewing the death certificates which accompanied some cases and independent medical evaluations which accompanied other cases, many claimants had nonoccupational illnesses which could account for increased interstitial markings radiographically. In many cases only a B reading was provided with no other clinical supporting data. Given the fact that there are well over 100 recognized causes for increase in interstitial markings, attribution to asbestos on the basis of the ILO report alone would overestimate the true incidence of asbestos related disease. Furthermore, many of these reports only indicate that the x-ray is "consistent" with asbestosis without making a definitive diagnosis of an asbestos related disease within reasonable medical probability or certainty.

The hazards of asbestos exposure are well described within the peer review literature as causing a broad spectrum of both benign and malignant disease involving the pleura and pulmonary parenchyma. In 1986 the American Thoracic Society adopted an official statement entitled, "The Diagnosis of NonMalignant Diseases Related to Asbestos"[18]. The committee opines it is necessary to have a history of exposure and latency and then identifies certain objective data which are of proven value in making the diagnosis of asbestosis including chest radiograph, a reduction in FVC, reduction in diffusion capacity and the presence of rales.

Of the diagnostic criteria, the committee considered the chest x-ray to be the most important. However, there are certain caveats given in regard to the x-ray. It is stated that given a clear history of exposure to asbestos, a diffuse interstitial fibrosis can be presumed to be due to asbestos as other forms of interstitial fibrosis are relatively uncommon. However, the next sentence states **"the prevalence of lesser degrees of interstitial fibrosis is not well known and considerable caution has to be exercised in attributing all such phenomena to asbestos exposure, either known or occult.".** The physician is advised that the diagnosis of asbestosis is a judgement based on careful consideration of **all** relevant clinical findings. The specificity of the above criteria increases with increasing numbers of the positive criteria which are found. Furthermore, it is stated "as in all clinical judgements, **confounding variables such as the presence of other clinical conditions** that affect this criteria should be evaluated".

In recent years, large numbers of claims alleging injury due to asbestos have been filed solely on the basis of ILO B readings. The overwhelming majority of such readings report the "lesser degrees of interstitial fibrosis" about which the ATS warns. The reader is referred to the ILO profusion chart cited earlier in this report. Only 2.5% of ILO reports were greater than a Level 1 on the ILO scale.

It is my understanding this ATS standard is no longer distributed by the ATS and is being revised. When published, the updated version should be reviewed in regard to any issue relevant to this study.

OCCUPATION

Occupation: In order to diagnose asbestosis, a history of non-trivial exposure and adequate latency are both required (ATS). In many of the claims submissions determination of such exposure was difficult. A sampling of occupations is included Exhibit (0-I).

In some cases, occupational history suggested other more probable causes than asbestos for the x-ray findings. The relative absence of pleural cases emphasizes the need to search for other causes of interstitial disease. Occupational history of sandblaster, foundry worker, steel mill worker, talc exposure, welder, molder, mold change, grinder, coal miner, etc. are examples.

In some cases, a word processed document appears to omit any description of occupation Exhibit (0-2). In another case Exhibit (0-3) employment of "chemist and worked in management" was reported to indicate "occasion to be heavily exposed to asbestos". Exposures such as this would warrant more detailed explanation. In other cases, reports diagnosing asbestosis are drafted to allow attachment of employer and date of employment, but without detailed history of exposure by the examiner Exhibit (0-4).

FUTURE CLAIMS

III. FUTURE CLAIMS:

A search of the peer review literature reveals that few have ever attempted to project the future incidence of asbestos related disease. No one has ever successfully predicted the future number of asbestos claims [17]. No one has ever attempted to predict the number of future impaired nonmalignant claims [26].

The number of future cases of nonmalignant asbestos disease with impairment as defined in the NSP Agreement should decline from present levels with passage of time based on our current knowledge and the peer review literature:

1) Asbestosis is a dose-response related disease measured in terms of cumulative exposure

   A) The greater the cumulative exposure, the more likely the disease is to occur

   B) The greater the cumulative exposure, the worse the injury (impairment) is likely to be

2) Significant declines in exposure occurred in the mid to late 1960s with significant further decline with the institution of OSHA regulation in the early to mid 1970s.

3) Risk of asbestosis peaks approximately 30-40 years after onset of exposure and declines thereafter. Therefore, the risk of asbestosis among those exposed prior to 1962 - 1972 has already begun to decline.

4) In 1978, Dr. Selikoff noted that because of reduced exposure new cases should be declining - especially interstitial disease while pleural changes may be less affected.

5) In 1986, the American Thoracic Society opined that asbestosis should be a vanishing disease based on exposure levels at that time [18].

6) Stayner, Dement, et al (NIOSH, CDC) opined a risk of 2/1000 for asbestosis at the current OSHA standard exposure for 45 years.

Further, they opine a "sublinear" exposure - response relationship for asbestosis - implying the risk for asbestosis drops off more rapidly with reduction in exposure than does the risk for cancer. It was their opinion that the risk of **lung cancer** was **higher** than the risk for asbestosis (5/1000 for cancer, 2/1000 for asbestosis). It was unclear if this refers to morbidity or mortality.

7) In 1982 Nicholson & Selikoff[25] opined deaths from asbestosis would only occur from those "exposed to **high** concentrations over **long periods** of time" and would largely occur in insulators, manufacturing workers and long term shipyard employees. They opined the number of asbestosis deaths would range from 1/3 to 3/4 the deaths from **mesothelioma among insulators**. They opined the number of deaths from asbestosis to be 1/3 of the mesothelioma deaths for shipyard workers and manufacturing sector. They noted 200 deaths per year (1982) and opined that number might double (400 deaths) for 2 decades **then decline** thereafter.

8) OSHA predicted a threshold of 10 fiber years below which asbestosis was unlikely to occur. At 10 fiber-years the rate of asbestosis was 5/1000 exposed workers. This assumes 0.2 fiber/cc exposure 40 hours a week for a 50 year working lifetime[20].

9) In 1983 Walker (Harvard School of Public Health Manville Study)[23] opined that between 2000-2004 there would only be **4,400 men alive with asbestosis** based on incidence of mesothelioma or **11,400** based on asbestosis and mesothelioma mortality rates. Walker opined "grade 1 asbestosis - were those minimally diseased as opposed to those "genuinely" diseased. He opined a "very indefinite" upper limit of the order of 120,000 asbestos claims from 1982 forward. He opined that the rate of "minimally diseased" cases "would continue to flow at a rate (determined by sociolegal factors) which is unlikely to be bounded by purely medical or epidemiological factors...."

10) Lilis and Selikoff studied maintenance workers in chemical plants and found "restrictive" defect in 5.4% [19]. Restrictive defect was defined as FVC <80% but does not define presence or absence of reduction of FEV1/FVC. Refinery workers were found to have restrictive defect in 14% but 10% may have also had obstruction.

11) Kilburn studied 12,856 men exposed to asbestos. Only 85 (2.5%) had 2/2 or greater. Only 52 had a restrictive defect (**0.4%**). "Men with radiographically advanced asbestosis had normal TLC (105.5% predicted) accompanied by airway obstruction. He described restrictive disease as "rare" (0.4%). Kilburn went on to opine that "radiographically advanced asbestosis and physiological restrictive disease were mutually exclusive." He further opined in 2000 that asbestosis was in its "downward leg".[22]

Opinion: Future Claims

This study strongly suggests that the current incidence of impaired nonmalignant cases is significantly **less** than the incidence of nonmalignant **claims**. In my opinion, it is from this baseline and under similar scrutiny that future "cases" should be gauged.

It is my opinion that in the future the number of cases showing impairment at NSP levels will decline from current levels:

1) Future cases (Oct .2000-2049) are likely to have far less exposure than claims filed in prior years. Those first exposed in the mid 1960s will have lower cumulative exposures than those first exposed in the 1940s and 1950s. There is not a rational basis for the incidence of asbestosis to be higher in the future than it was in the past.

2) The "pool" of those at future risk should already be significantly reduced by the approximately 500,000 claims previously filed against O.C.

3) Further reduction in claims should occur as those with significant prior exposure in the 1940s, 50s, and early 60s have now reached 40 years since first exposure and their future risk of nonmalignant disease drops .based on the works of Selikoff, Dement and others.

4) Many of those with higher levels of exposure in the 1940s, 50s and early 60s have either reached average life expectancy for a male (74.5 years) or will soon be approaching that age where other risk factors for mortality are commonly encountered.

5) Not only should the incidence of nonmalignant disease decline, but the incidence of severe disease accompanied by impairment as defined under the terms of the Agreement should become "rare" as already noted by more recent authors.

6) Many of the current cases under study do **not** represent a diagnosis of nonmalignant asbestos disease. Rather terminology such as "consistent with" or "in the proper clinical setting" is noted. Requiring a diagnosis of asbestosis based on published ATS criteria, review of radiographs by a random selection of reliable B readers or physicians familiar with the ILO and utilization of all ATS criteria for PFT performance (including predicted values) will help focus on those truly impaired. This should reduce the number of marginal claims in the future.

Conclusion